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Self-reported health and cardiovascular reactions to psychological stress in a large community sample: Cross-sectional and prospective associations

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Abstract

Exaggerated cardiovascular reactions to acute psychological stress have been implicated in a number of adverse health outcomes. This study examined, in a large community sample, the cross-sectional and prospective associations between reactivity and self-reported health. Blood pressure and heart rate were measured at rest and in response to an arithmetic stress task. Self-reported health was assessed concurrently and five years later. In cross-sectional analyses, those with excellent/good self-reported health exhibited larger cardiovascular reactions than those with fair/poor subjective health. In prospective analyses, participants who had larger cardiovascular reactions to stress were more likely to report excellent/good health five years later, taking into account their reported health status at the earlier assessment. The findings suggest that greater cardiovascular reactivity may not always be associated with negative health outcomes.

Descriptors: Blood pressure, Heart rate, Acute psychological stress, Self-reported health

The reactivity hypothesis considers that exaggerated cardiovascular reactions to acute psychological stress are a risk factor for cardiovascular pathology (Lovallo & Gerin, 2003; Schwartz, et al., 2003). In support of this, several prospective studies have now shown with reasonable consistency that high reactivity confers an additional risk for a range of cardiovascular outcomes, including high blood pressure, carotid atherosclerosis, carotid intima thickness, and increased left ventricular mass (e.g. Allen, Matthews, & Sherman, 1997; Barnett, Spence, Manuck, & Jennings, 1997; Carroll, Ring, Hunt, Ford, & Macintyre, 2003; Kamarck, et al., 1997; Lynch, Everson, Kaplan, Salonen, & Salonen, 1998; Markovitz, Raczynski, Wallace, Chettur, & Chesney, 1998; Treiber, et al., 2003).

The impact of cardiovascular reactivity on other health outcomes has attracted less attention. However, studies have examined the association between reactivity and obesity and depression, the rationale being that both have been identified as risk factors for cardiovascular disease outcomes. Adiposity has been consistently linked to cardiovascular disease morbidity (Hirani, Zaninotto, & Primatesta, 2007; Mokdad, et al., 2003) and mortality (Adams, et al., 2006; Allison, Fontaine, Manson, Stevens, & VanItallie, 1999; Calle, Thun, Petrelli, Rodriguez, & Heath, 1999; Stevens, et al., 1998). Greater adiposity has also been found to be associated with higher reactivity in some (Davis, Twamley, Hamilton, & Swan, 1999; Goldbacher, Matthews, & Salomon, 2005; Waldstein, Burns, Toth, & Poehlman, 1999), although it should be conceded not all, studies (Carroll, Phillips, & Der, 2008). Depression has also been linked prospectively to all-cause mortality and to cardiovascular disease mortality in particular (for reviews, see (Hemingway & Marmot, 1999; Wulsin, Vaillant, & Wells, 1999). There is also tentative evidence that higher depressive symptomatology is associated with greater cardiovascular reactivity (Kibler & Ma, 2004). This sort of inquiry embraces the possibility that excessive cardiovascular reactions to acute stress exposure may be implicated in a much broader range of health outcomes than originally envisaged by the reactivity hypothesis.

If reactivity has wider implications for health, it is perhaps curious that no study that we know of has examined whether exaggerated cardiovascular reactions to acute stress are associated with poor self-reported health. Intuitively, self-reported health is an interesting candidate in this context. The results of numerous large-scale prospective epidemiological studies testify that self-reported health predicts various health outcomes including mortality in a dose-response fashion, independently of traditional risk factors and medical status; those reporting poor health have a mortality risk two to seven times greater than those reporting excellent health (for review, see e.g.(Idler & Benyamini, 1997). If self-reported health is affected by cardiovascular morbidity and its precursory processes, it might be expected to be related to reactivity. The present analyses, then, examined the relationship between cardiovascular reactions to an acute psychological stress task and self-reported health both cross-sectionally and prospectively in a large community sample.

Given the evidence linking reactivity to cardiovascular outcomes, it was hypothesized that large magnitude reactions would be more characteristic of those reporting relatively poor health. In essence, this might be regarded as an important test of the scope and the limitations of the reactivity hypothesis.

Method

Participants

Data were collected as part of the West of Scotland Twenty-07 Study. Participants were all from Glasgow and surrounding areas in Scotland, and have been followed up at regular intervals since the baseline survey in 1987 (Ford, Ecob, Hunt, Macintyre, & West, 1994). The study's principle aim was to investigate the processes that produce and maintain socio-demographic differences in health (Macintyre, 1987). Participants were chosen randomly with probability proportional to the overall population of the same age within a post code area (Ecob, l987). Thus, this is a clustered random stratified sample. Three narrow age cohorts were chosen (aged 15, 35, and 55 years at entry), each reflecting important stages of life and transitions, and so that age specific effects could be estimated with greater precision than an all age sample of the same size. The data reported here are from the third and fourth follow-ups. The mean (SD) temporal lag between the two follow-ups was 5.5 (1.00) years.

At the third follow-up, cardiovascular reactions to an acute psychological stress task were measured (Carroll, et al., 2000; Carroll, Ring, Hunt, Ford, & Macintyre, 2003). Self-reported health data were available at second, third, and fourth follow-ups, but as we were interested in the cross-sectional and prospective association between cardiovascular reactivity and self-reported health, we focused on the self-reported health data from the third and fourth follow-ups. At the third follow-up, full data for cardiovascular variables and self-reported health were available for 1647 participants. At the fourth follow-up, self-reported health data were available for 1318 participants, an attrition rate of 20%. The sample comprised three distinct age cohorts: 592 (36%) 24-year olds, 624 (38%) 44-year olds, and 431 (26%) 63-year olds. The overall mean age at the third follow-up was 41.8 (SD = 15.44) years. The exact mean (SD) ages of the young, middle aged, and eldest cohorts were 23.7 (0.56) 44.1 (0.85), and 63.1 (0.67) years, respectively. There were 890 (54%) women and 757 (46%) men in the sample, with 772 (47%) from manual and 870(53%) from non-manual occupation households. Household occupational group data were not available for five participants. The sample was almost entirely Caucasian, reflecting the West-of-Scotland population from which it was drawn. Mean (SD) body mass index, calculated from measured height and weight was 26.7 (4.26) kg/m^2. The study was approved by the appropriate Ethics committees.

Apparatus and procedure

Participants were interviewed and tested in a quiet room in their homes by trained nurses. At the third follow-up, the interview assessed demographic, health behaviour, and psychosocial variables such as life events. Household occupational group was classified as manual or non-manual from the occupation of the head of household, using the Registrar General's classification of occupations (1980). Head of household was usually the man. They then undertook an acute psychological stress task: the paced auditory serial addition test (PASAT), which has been shown in numerous studies to reliably perturb the cardiovascular system (Ring, Burns, & Carroll, 2002; Winzer, et al., 1999) and to demonstrate good test-retest reliability (Willemsen, et al., 1998). Participants were presented with a series of single digit numbers by audiotape and requested to add sequential number pairs while retaining the second of the pair in memory for addition to the next number presented, and so on throughout the series. Answers were given orally and, if participants faltered, they were instructed to recommence with the next number pair. The correctness of answers was recorded as a measure of performance. The first sequence of 30 numbers was presented at a rate of one every four seconds, and the second sequence of 30 at one every two seconds. The whole task took three minutes, two minutes for the slower sequence and one minute for the faster sequence. A brief practice was given to ensure that participants understood the requirements of the task. Only those who registered a score on the PASAT were included in the analyses. Out of a possible score of 60, the mean score was 40.9 (SD = 9.03).

Systolic blood pressure (SBP), diastolic blood pressure (DBP) and heart rate (HR) were measured by an Omron (model 705CP) sphygmomanometer. This semi-automatic blood pressure device is recommended by the European Society of Hypertension (O'Brien, Waeber, Parati, Staessen, & Myers, 2001). Following the interview, (at least an hour), there was then a formal 5-minute period of relaxed sitting, at the end of which a resting baseline reading of SBP, DBP, and HR was taken. Task instructions were then given, followed by the brief practice. Two further SBP, DBP, and HR readings were taken during the task, the first initiated 20 seconds into the task (during the slower sequence of numbers), and the second initiated 110 seconds later (at the same point during the faster sequence). For all readings, the nurses ensured that the participant's elbow and forearm rested comfortably on a table at heart level. The two task readings were averaged and the resting baseline value subsequently subtracted from the resultant average task value to yield reactivity measures for SBP, DBP, and HR for each participant. Data were available for self-reported health at both time points. Participants were presented with the question, 'Would you say that for someone your age your own health is…' and given four response options: excellent, good, fair, poor. The self-reported health data were negatively skewed because only a very small proportion (N=90 at the third follow-up and N=72 at the fourth follow-up) of participants reported

poor health, with most (80% and 75%) reporting either fair or good health. Consequently, for the present analyses, the excellent and good categories were collapsed, as were the fair and poor categories, to yield a simple binary variable.

Statistical analyses

Repeated measures ANOVAs, using baseline and task values, were undertaken to confirm that the PASAT perturbed cardiovascular activity and to compare variations in SBP, DBP, and HR reactivity between age cohorts, sexes, and household occupational groups. To examine the socio-demographic patterning of self-reported health, χ^2 was applied. Prior to testing the association between self-reported health and reactivity, the relationship between health and baseline cardiovascular level was examined using ANOVA. Cross-sectional analyses of reactivity (the difference between task and baseline values) were by ANCOVA, with SBP, DBP, and HR reactivity as separate continuous variables and self-reported health status at the third follow-up as the grouping variable; baseline cardiovascular level was entered as a covariate. Further ANCOVAs were then undertaken that, in addition to baseline cardiovascular level, adjusted for socio-demographic characteristics and other potential confounders: age cohort, sex, occupational group, PASAT performance score, body mass index, and antihypertensive medication status. These potential confounding variables were selected on the basis of their previously reported associations with reactivity in this sample (Carroll, et al., 2000; Carroll, Phillips, & Der, 2008). Slight variations in degrees of freedom reflect occasional missing data for some variables. For both ANOVAs and ANCOVAs, partial η^2 is reported as a measure of effect size. Prospective analyses examining the association between reactivity and subsequent self-reported health status was by logistic regression, with self reported health at the fourth follow-up as the dependent variable and reactivity as the independent variable; these initial analyses adjusted for self-reported health status at the third follow-up. Further analyses were then conducted adjusting for the other potential confounders.

Results

Cardiovascular reactivity

The stress task provoked significant increases in SBP, $F(1,1630) = 1535.21$, $p < .001$, $\eta^2_p = .485$, DBP, $F(1,1630) = 1017.31$, $p < .001$, $\eta^2_p = .384$, and HR, $F(1,1630) = 1068.26$, $p < .001$, $\eta^2_p = .396$. As can be seen from Table 1, HR reactivity declined with age, $F(2,1630) = 19.49$, $p < .001$, $\eta^2_p = .023$; with the youngest cohort exhibiting higher reactivity than the middle cohort who, in turn, showed higher reactivity than the eldest cohort ($p < .05$ in each case). Heart rate reactivity was also greater in men, $F(1,1630) = 5.63$, $p = .02$, $\eta^2_p = .003$, and in participants from non-manual occupational status households, $F(1,1630) = 16.96$, $p < .001$, $\eta^2_p = .010$. SBP reactivity varied

significantly among the age cohorts, $F(2,1630) = 7.03$, $p = .001$, $\eta^2_p = .009$, with the youngest cohort having significantly lower reactivity than the other two cohorts ($p < .05$ in both cases). Women had smaller SBP reactions than men, $F(1,1630) = 21.24$, $p < .001$, $\eta^2_p = .013$. DBP reactivity did not vary significantly with age cohort or sex but those non-manual households exhibited larger DBP increases, $F(1,1630) = 5.25$, $p = .02$, $\eta^2_p = .003$. There were few interaction effects of note. For SBP, there was an age cohort × sex interaction, $F(1,1630) = 7.05$, $p = .001$, $\eta^2_p = .009$; larger SBP reactions for men were evident only in the eldest cohort. For DBP, the age cohort × occupational status interaction just met the criterion for statistical significance, $F(1,1630) = 3.05$, $p = .05$, $\eta^2_p = .004$; the difference in DBP reactivity between household occupational groups occurred only for the eldest cohort.

[Insert Table 1 about here]

Self-reported health

Overall, 1211 (74%) and 967 (73%) of the participants reported that they enjoyed relatively good or excellent health at the third and fourth follow-ups respectively. For 134 (10%) of the sample, their self-reported health improved between follow-ups, whereas for 176 (13%) it deteriorated. At the third follow-up, proportionally more of those from manual (30%) compared to non-manual occupational households (23%) reported poor or only fair health, $\chi^2(1) = 11.78$, $p = .001$. This difference between manual and non-manual occupational groups was preserved at the fourth follow-up, $\chi^2(1) = 17.02$, $p < .001$. There were no sex differences in self-reported health at either follow-up and significant variation with age emerged only at the third follow-up, where, compared to the other two cohorts (30%), proportionally less of the middle cohort (20%) reported relatively poor or only fair health, $\chi^2(1) = 18.34$, $p < .001$.

Self-reported health and baseline cardiovascular activity

Those reporting relatively good health at the third follow-up had slightly higher baseline SBP, $F(1,1642) = 3.48$, $p = .06$, $\eta^2_p = .002$, and DBP, $F(1,1642) = 3.99$, $p = .05$, $\eta^2_p = .002$; the respective mean (SD) values for the good and poor health status groups were 129.6 (20.20) and 79.12 (11.33) mmHg and 127.5 (20.98) and 77.9 (12.25). In contrast the better self-reported health group had significantly lower baseline HR, $F(1,1642) = 5.62$, $p = .02$, $\eta^2_p = .003$; the mean (SD) values for the good and poor health status groups were 66.4 (10.91) and 67.8 (10.40) beats per minute, respectively. ANCOVA was applied to explore the association between baseline cardiovascular activity and self-reported health status at the fourth follow-up, adjusting for health status at the third follow-up. The only effect to emerge was for HR, $F(1,1313) = 9.26$, $p = .002$, η^2_p

= .007; the respective mean (SD) values for the good and poor health status group were 65.8 (10.46) and 68.4 (11.20). This association was explored further using logistic regression, in which self-reported health status at the fourth follow-up was the dependent variable, baseline HR the independent variable, and health status at the third follow-up the covariate. Baseline HR predicted self-reported health at the fourth follow-up in this model, OR = .98, 95%CI .97 - .99, p = .002; those with higher baseline HR were more likely to show a negative shift over time in self-reported health. Finally, baseline cardiovascular activity correlated negatively with reactivity: for SBP, r (1645) = -.19, $p < .001$, for DBP, r (1645) = -.29, $p < .001$, and for HR, r (1645) = -.29, $p < .001$.

Self-reported health and cardiovascular reactivity: Cross-sectional analyses

In ANCOVA, adjusting for baseline, participants who reported relatively excellent or good health at the third follow-up had larger SBP, $F(1,1641) = 8.01$, $p = .005$, $\eta^2_p = .005$, DBP, $F(1,1641) = 7.19$, $p = .007$, $\eta^2_p = .004$, and HR, $F(1,1641) = 6.16$, $p = .01$, $\eta^2_p = .004$, reactions than those who reported poor or only fair health. The mean (SD) reactivity values for the two groups are presented in Figure 1. These effects were re-examined adjusting for a number of additional potential confounding variables: age cohort, sex, occupational group, PASAT performance score, body mass index, and antihypertensive medication status. The effect of health status group on reactivity remained significant for SBP, $F(1,1627) = 5.45$, $p = .02$, $\eta^2_p = .003$, and DBP, $F(1,1627) = 6.53$, $p = .01$, $\eta^2_p = .004$, but not for HR, $F(1,1627) = 2.03$, $p = .16$, $\eta^2_p = .001$, reactivity. Supplementary ANCOVAs, where the covariates were entered one at a time, indicated that it was the PASAT performance score that most attenuated the association between self-reported health and HR reactivity.

[Insert Figure 1 about here]

Self-reported health and cardiovascular reactivity: Prospective analyses

In logistic regression analyses, in which self-reported health status at the third follow-up was entered as a covariate, cardiovascular reactivity predicted health status five years later at the fourth follow-up: for SBP reactivity, OR = 1.01, 95%CI 1.00 – 1.03, p = .02, for DBP and HR reactivity, OR = 1.02, 95%CI 1.01 – 1.04, p = .002. Thus, participants who exhibited relatively high cardiovascular reactions to stress reported better self-reported health five years later independently of health status at the earlier time point. These associations are illustrated in Figure 2, which presents the estimated mean cardiovascular reactions for those reporting excellent/good or fair/poor health at the fourth follow-up, controlling for self-reported health status at the third follow-up. Finally, logistic regressions were computed in which, in addition to health status at the third follow-

up, the other covariates (above) were entered. The positive associations between DBP, OR = 1.02, 95%CI 1.00 – 1.04, p = .006, and HR, OR = 1.02, 95%CI 1.00 – 1.03, p = .02, reactivity and self-reported health status between follow-ups were still evident, although the relationship with SBP reactivity was no longer significant, OR = 1.01, 95%CI 0.99 – 1.02, p = .09.

[Insert Figure 2 about here]

In order to illustrate these effects further, we then compared those in the top and bottom quartiles of cardiovascular reactivity in terms of future self-reported health status. The means (SD) for the top and bottom quartiles of reactivity were: SBP, 27.0 (7.25) and -2.7 (6.14) mmHg; DBP, 18.4 (6.76) and -2.2 (5.29) mmHg; and HR, 19.1 (7.50) and -3.0 (5.73) bpm. Those in the top quartile of reactivity were more likely to report good health five years later than those in the bottom quartile: for SBP, OR = 1.15, 95%CI 1.02 – 1.29, p = .02, DBP, OR = 1.23, 95%CI 1.04 – 1.45, p = .02, and HR reactivity, OR = 1.20, 95%CI 1.08 – 1.34, p = .001.

Sensitivity analyses

The main cross-sectional and prospective analyses were repeated using the four category versions of the self-reported health variables. These yielded virtually identical outcomes. In ANCOVA, adjusting for baseline, there was a main effect of self-reported health at the third follow-up on SBP, $F(3,1639) = 5.11$, $p = .002$, $\eta^2_p = .009$, DBP, $F(3,1639) = 2.84$, $p = .04$, $\eta^2_p = .005$, and HR, $F(3,1639) = 2.86$, $p = .04$, $\eta^2_p = .005$, reactions; those with higher reactivity reported better health. The means (SD) are reported in Table 2, which indicates an orderly positive relationship between reactivity and self-reported health status. For the prospective analyses, we used multinomial logistic regression, entering reactivity as a continuous variable and adjusting for self-reported health status at the third follow-up. Likelihood ratio tests indicated a significant association between reactivity and self-reported health at the fourth follow-up: for SBP, $\chi^2(3) = 12.26$, p = .007; for DBP, $\chi^2(3) = 10.51$, p = .02, for HR, $\chi^2(3) = 11.33$, p = .01.

[Insert Table 2 about here]

Discussion

The majority of participants in the present study reported excellent/good health. The percentages with relatively poor self-reported health at the two time points are in accordance with those observed elsewhere. For example, the proportion of American men and women admitting to only fair or poor health in 2005 was reported to be 26 and 27 per cent (Kramarow, Lubitz, Lentzner, &

Gorina, 2007). However, contrary to our initial expectations, self-reported health in a large community sample was positively related to cardiovascular reactivity in cross-sectional analyses. Participants reporting good or excellent health exhibited larger blood pressure and heart rate reactions to an acute stress exposure than those reporting only fair or poor health. In contrast to many reactivity studies, the richness of the current database allowed us to control for a number of candidate confounding variables, and all our analyses adjusted for baseline cardiovascular levels. The significant association between self-reported health and SBP and DBP reactivity survived additional adjustment for medication likely to affect blood pressure and heart rate, body mass index, performance score on the stress task, and demographic variables, age, sex, and occupational status. It should be conceded, though, that the effect sizes were somewhat attenuated. For HR reactivity, however, the association with self-reported health status was no longer statistically significant in the fully adjusted ANCOVA model. These outcomes underscore the need to take into account potential confounders in observational studies. It might be argued that the statistically robust associations seen for SBP and DBP reactivity could still be due to residual confounding. The ability of covariance analyses to adjust fully for some putative confounder depends on its accuracy of measurement; the greater the accuracy of measurement of the covariate, the greater the confidence that it will be fully accounted for in the statistical model (Christenfeld, Sloan, Carroll, & Greenland, 2004). We would argue, however, that most of the covariates, such as age, sex, body mass index, and task performance, included in our analyses were measured accurately.

The prospective analyses revealed small but statistically significant associations between reactivity and future self-reported health status; participants who had larger cardiovascular reactions to stress were more likely to report excellent/good health at the later follow-up, taking into account their reported health status at the earlier assessment. This was again contrary to initial expectations, but consistent with the outcomes of the cross-sectional analyses. In addition, the associations between DBP and HR, but not SBP, reactivity and future self-reported health status remained significant in the fully adjusted model.

The reactivity hypothesis originally postulated that exaggerated cardiovascular reactions to stress contributed to the development of high blood pressure and, by extension, related cardiovascular disease outcomes (Lovallo & Gerin, 2003; Schwartz, et al., 2003). However, the hypothesis has occasionally had to shoulder additional explanatory burdens. Several of the risk factors for cardiovascular disease have been hypothesised to exert their effects, at least in part, through an association with heightened reactivity. For example, depression has been linked prospectively to cardiovascular disease morbidity and mortality and hypothesised to take its toll through its effects on cardiovascular reactions to stress (Kibler & Ma, 2004). A meta-analysis of 11 relevant studies found small to moderate, although not statistically significant, effects indicative of

a positive relationship between depressive symptomatology and cardiovascular reactions to acute psychological stress (Kibler & Ma, 2004). In contrast, though, two recent substantial analyses have found that symptoms of depression were negatively associated with reactivity; the greater the symptoms of depression, the lower the reactivity (Carroll, Phillips, Hunt, & Der, 2007; York, et al., 2007). Similarly, obesity and adiposity have also been proposed to affect cardiovascular disease through heightened reactivity (Davis, Twamley, Hamilton, & Swan, 1999; Waldstein, Burns, Toth, & Poehlman, 1999), but we have recently found that obesity was associated with lower HR reactivity both cross-sectionally and prospectively (Carroll, Phillips, & Der, 2008). Further, low socioeconomic position is a well documented risk for cardiovascular disease and exaggerated cardiovascular reactions to stress have again been suggested as a possible pathway (Strike & Steptoe, 2004). However, in this dataset (Carroll, Ring, Hunt, Ford, & Macintyre, 2003) and in another large population study (Carroll, Davey Smith, Sheffield, Shipley, & Marmot, 1997), low socioeconomic position was associated with relatively low reactivity. Finally, relatively low cardiovascular reactivity has been found among current smokers (Roy, Steptoe, & Kirschbaum, 1994). It would appear that hypertension and other cardiovascular disease manifestations aside, high cardiovascular reactivity may not always be associated with negative health outcomes and behaviours, given that there is now, at least preliminary, evidence that it may characterise those from higher socio-economic positions, those with fewer symptoms of depression, those who are leaner, and those who do not smoke. We can now tentatively add better self-reported health to this list of virtuous correlates.

There are precedents for positive associations between acute stress exposure and beneficial health outcomes. Acute exposure to moderate behavioural challenges has been associated with better health, mainly for immunological outcomes, and increased longevity in a number of recent laboratory animal studies (Arumugam, Gleichmann, Tang, & Mattson, 2006; Shevchuk & Radoja, 2007). This phenomenon is termed hormesis. Direct evidence in humans, however, is sparse (Leslie, 2005). Nevertheless, our group has shown that exposure to a longer version of the mental stress task used in the present study was associated with an enhanced antibody response to the influenza vaccine in women (Edwards, et al., 2006). In addition, the magnitude of the interleukin-6 response to the stress in women was positively associated with the magnitude of the antibody response. The present results may be tentatively regarded, then, as further human evidence of hormesis.

The present study is not without limitations. First, it should be acknowledged that the effect sizes in our prospective analyses are small, when reactivity is treated as a continuous variable. However, our effects are of the same order as the positive associations between cardiovascular reactivity and future blood pressure status in this sample (Carroll, Ring, Hunt, Ford, & Macintyre,

2003) and others (Carroll, Smith, Sheffield, Shipley, & Marmot, 1995; Carroll, et al., 2001; Markovitz, Raczynski, Wallace, Chettur, & Chesney, 1998; Matthews, Woodall, & Allen, 1993; Newman, McGarvey, & Steele, 1999). In addition, these effect sizes in the categorical analyses are similar to, for example, those linking BMI, as a categorical variable, and future coronary heart disease risk (see e.g. Bogers, et al., 2007), which is considered to be a clinically significant association. Second, we measured only blood pressure and HR. It could have proved instructive to have the sort of comprehensive assessment of haemodynamics afforded by impedance cardiography. However, the decision to test participants in their own homes and the size of the sample precluded the use of impedance cardiography. Third, determining causality is impossible from cross-sectional study and still fraught with pitfalls in prospective analyses (Christenfeld, Sloan, Carroll, & Greenland, 2004). However, we did adjust statistically for a broad range of potential confounders. Nevertheless, residual confounding as a consequence of poorly measured or un-measured variables cannot be wholly discounted. For example, one very parsimonious explanation for the direction of relationship observed in the present study is that participants with poorer subjective health were relatively de-motivated and simply engaged less with the stress task. This would be reflected in a poorer stress task performance score and participants who reported, at the third follow-up, that their health was only fair or poor, mean = 41.9, SD = 9.69, performed less well than those reporting excellent/good, mean = 44.5, SD = 8.89, health, $F(1, 1642) = 24.57$, $p < .001$, $\eta^2_p = .015$. However, the majority of both the cross-sectional and prospective associations remained significant after adjustment for performance. Fourth, and relatedly, we had to rely on performance score as our measure of task engagement and a more specific measure of effort would have provided a stronger test of possible confounding. Finally, we employed only a single item measure of self-reported health. However, single item assessments of subjective health status with these four response options are very much the norm and have been successfully used to provide independent prospective prediction of all-cause mortality in a number of epidemiological studies, such as the Longitudinal Study of Aging (Grant, Piotrowski, & Chappell, 1995), the National Health and Nutrition Examination Survey (Idler, Russell, & Davis, 2000), and the Yale Health and Aging Project (Idler, Kasl, & Lemke, 1990).

This is the first study we know of to examine the association between cardiovascular reactions to acute psychological stress and self-reported health. Contrary to our initial expectations, participants with better the self-reported health exhibited greater cardiovascular reactivity. In prospective analyses, higher reactivity was associated with better subjective health five years, controlling for self-reported health at earlier time point. The hypothesis that high cardiovascular reactivity is injurious to health may apply only to a particular set of health outcomes. Further, considered along with recent findings on depression, obesity, smoking, and socio-economic status,

our results suggest that higher reactivity may not always be associated with negative health outcomes and behaviours.

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Table 1. Mean (SD) SBP, DBP, and HR baseline and reactivity by age cohort, sex, and occupational status

	SBP		*DBP*		*HR*	
	Baseline	*Reactivity*	*Baseline*	*Reactivity*	*Baseline*	*Reactivity*
Age Cohort:						
Youngest (N = 592)	120.0 (15.07)	10.1 (10.24)	73.4 (10.08)	6.8 (9.04)	67.5 (11.00)	10.0 (10.56)
Middle (N = 624)	127.1 (18.08)	12.3 (11.44)	80.6 (11.13)	7.1 (8.03)	66.7 (11.17)	7.7 (10.00)
Eldest (N = 431)	144.4 (21.68)	12.3 (13.92)	83.8 (11.17)	7.0 (8.92)	65.7 (9.92)	6.1 (7.74)
Sex:						
Male (N = 757)	134.7 (18.25)	12.8 (11.77)	81.2 (11.18)	7.2 (8.43)	64.7 (10.43)	8.7 (9.73)
Female (N = 890)	124.3 (21.07)	10.4 (11.70)	76.8 (11.56)	6.8 (8.81)	68.4 (10.84)	7.6 (9.83)
Occupational Group:						
Manual (N = 772)	130.5 (21.44)	11.1 (12.22)	79.3 (11.93)	6.5 (9.07)	67.0 (11.26)	6.9 (9.54)
Non-manual (N = 872)	127.8 (19.58)	11.8 (11.39)	78.4 (11.29)	7.3 (8.24)	66.5 (10.40)	9.1 (9.90)

Table 2: Means (SD) for cardiovascular reactivity at each level of self-reported health at the third follow-up

Self-reported Health	SBP	DBP	HR
Poor	8.0 (10.30)	5.6 (9.67)	6.4 (8.69)
Fair	11.0 (11.90)	6.4 (8.74)	7.0 (9.51)
Good	11.6 (11.77)	7.1 (8.45)	8.3 (9.90)
Excellent	13.1 (11.58)	7.7 (8.62)	9.5 (9.92)

Figure 1: Estimated marginal mean (SE) cardiovascular reactivity, adjusting for baseline level, for those reporting excellent/good or fair/poor health at the third follow-up.

Figure 2: Estimated marginal mean (SE) cardiovascular reactivity for those reporting excellent/good or fair/poor health at the fourth follow-up, adjusting for health status at the third follow-up.